1,2-Epoxybutane (EBU); CASRN 106-88-7

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR EBU

File First On-Line 05/01/1992

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	not evaluated	
Inhalation RfC (I.B.)	yes	05/01/1992
Carcinogenicity Assessment (II.)	not evaluated	

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — 1,2-Epoxybutane (EBU)
CASRN — 106-88-7

Not available at this time.

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — 1,2-Epoxybutane (EBU)
CASRN — 106-88-7
Last Revised — 05/01/1992

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. Inhalation RfC Summary

Critical Effect	Exposures*	UF	MF	RfC
Degenerative lesions of the nasal cavity **2-Year Mouse Inhalation Study** **NTP, 1988**	NOAEL: None LOAEL: 147 mg/cu.m (50 ppm) LOAEL(ADJ): 26 mg/cu.m LOAEL(HEC): 4.8 mg/cu.m	300	1	2E-2 mg/cu.m

*Conversion Factors: MW = 72.12. Assuming 25 C and 760 mmHg, LOAEL (mg/cu.m) = 50 ppm x 72.12/24.45 = 147 mg/cu.m. LOAEL(ADJ) = 147 x 6 hours/24 hours x 5 days/7 days = 26 mg/cu.m. The LOAEL(HEC) was calculated for a gas:respiratory effect in the extrathoracic region. MVa = 0.06 cu.m/day, MVh = 20 cu.m/day, Sa (ET) = 2.9 sq. cm., Sh (ET) = 177 sq. cm. RGDR(ET) = (Mva/Sa) / (Mvh/Sh) = 0.183. LOAEL(HEC) = LOAEL(ADJ) x RGDR = 4.8 mg/cu.m.

I.B.2. Principal and Supporting Studies (Inhalation RfC)

NTP (National Toxicology Program). 1988. Toxicology and carcinogenesis studies of 1,2-epoxybutane (CAS No. 106-88-7) in F344/N rats and B6C3F1 mice (inhalation studies). ISS NTP-TR-329, NIH/PUB-88-2585.

F344/N rats (50/sex) were exposed to 0, 200, or 400 ppm 1,2-epoxybutane (EBU) (0, 590, or 1180 mg/cu.m; duration-adjusted concentrations were 0, 105, or 210 mg/cu.m) 6 hours/day, 5 days/week for 2 years. B6C3F1 mice (50/sex) were exposed to 0, 50, or 100 ppm EBU (0, 590, or 1180 mg/cu.m; duration- adjusted concentrations were 0, 26, or 52 mg/cu.m) under the same exposure regime. The purity of the EBU was reported as more than 99% (NTP, 1988; also reported in Dunnick et al., 1988). The animals were observed twice daily, weighed weekly for 13 weeks and monthly thereafter, and subjected to a clinical examination once a month. Necropsy and histological examination of approximately 30 tissues were performed on all animals. Survival was adversely affected in both male and female rats by both concentrations of EBU. In the mice, survival was adversely affected by the 100-ppm concentration in both sexes; only 9/50 female mice exposed to 100 ppm EBU survived until study termination. In mice exposed to 100 ppm, body weight gain was reduced significantly (more than 10%). Relative to control values, a 10-14% (male) and a 13-23% (female) decrease in body weight was noted. Female mice exposed to the lower concentration suffered a 12-16% decrease. Increases in the incidence of both granulocytic hyperplasia and splenic hematopoiesis were observed in female mice in a concentration-dependent manner. The incidence for both these lesions at the lower concentration was increased over controls. This effect was limited to female mice and did not occur in male mice or in male or female rats. These effects could be secondary to the inflammatory processes in the nasal cavity. Blood effects would be an extrarespiratory effect, and the LOAEL(HEC) would be 26 mg/cu.m. Blood effects were also reported in mice and rats in the study of Miller et al. (1981) discussed following. Significant increases in the incidence of nonneoplastic degenerative and proliferative lesions of the nasal cavity were observed in male and female rats exposed to both concentrations of EBU. Concentration- related increases in the incidence rate were noted for inflammation, epithelial hyperplasia of the respiratory epithelium, atrophy of the olfactory sensory epithelium, and hyperostosis of the nasal turbinate bone. Similarly, significant increases in the incidence of non-neoplastic lesions of the nasal cavity of both sexes of mice were documented. Concentration-related increases in the incidence of chronic inflammation, epithelial hyperplasia, and erosion (occurring in 23 of 99 mice at the lower concentration) were noted in exposed mice, including suppurative inflammation, epithelial hyperplasia, and erosion. For rats, this study indicates a LOAEL for effects in the nasal tract of 200 ppm [the corresponding LOAEL(HEC) = 19 mg/cu.m]. For mice, 50 ppm is a LOAEL based on the lesions in the upper respiratory tract [LOAEL(HEC) = 4.8 mg/cu.m]. No NOAEL could be derived from this study for either species.

I.B.3. Uncertainty and Modifying Factors (Inhalation RfC)

UF — The uncertainty factor of 300 reflects factors of 10 to protect unusually sensitive individuals, 10 for the use of a LOAEL rather than a NOAEL, and 3 for interspecies extrapolation. An additional factor for lack of developmental/reproductive effects is not deemed necessary as the results from the chronic NTP study, in concert with chronic studies of epoxy analogs of EBU, indicate extrarespiratory circulation of EBU to be minimal.

MF — None

I.B.4. Additional Studies/Comments (Inhalation RfC)

Male and female B6C3F1 mice (10/sex/group) were exposed to greater than 99% pure EBU at the following concentrations for a total of 13 weeks: 0, 50, 100, 200, 400, or 800 ppm (0, 147, 295, 590, 1180, or 2360 mg/cu.m) (NTP, 1988). Based on exposure for 6 hours/day, 5 days/week, the corresponding duration-adjusted values are 0, 26, 53, 105, 211, or 421 mg/cu.m. The vapor was generated from the heated liquid and pumped directly into the exposure chamber. The animals were observed twice daily and weighed weekly. Histopathological examinations were performed on approximately 30 tissues from control animals and animals from the two highest concentration groups except in the case of the nasal turbinates which were examined in all animals at all exposure levels. NTP procedures were followed in the preparation and examination of all tissues. All mice exposed to 800 ppm and 2 of 10 males exposed to 50 ppm died, although no mortality was noted at any other exposure level. No significant weight depression was observed in any exposure group compared to controls. Systemic histopathology, which occurred only in the mice at the highest exposure level, included atrophy and necrosis of the spleen and thymus, and renal necrosis. The principal tissue affected at the highest concentration was the nasal passage. These tissues were necrotic in 15 of 20 animals at the highest exposure with less severe effects occurring at lower concentrations. Acute inflammation of the turbinates was observed in a concentration-related manner in 14/20 animals at 800 ppm and in 5/20 at 400 ppm; none was observed at lower concentrations. A lesser grade of inflammation, serous, was also observed in a concentration-related manner at lower concentrations in 20 of 20 mice exposed to 200 ppm, in 7/20 mice (all females) at 100 ppm, but not in any of the animals exposed to 50 ppm or the controls. The NOAEL for these effects is 50 ppm. As the serous inflammation was noted exclusively in female mice at 100 ppm, the LOAEL, the HEC is calculated based on default female ventilatory parameters [NOAEL(HEC) = 3.2 mg/cu.m, LOAEL(HEC) = 6.5 mg/cu.m]. Although this study identifies a NOAEL at 50 ppm, it should be noted that this level (50 ppm) produced severe nasal effects in mice in the chronic study discussed above (NTP, 1988), precluding use of this study in deriving an RfC.

Male and female F344/N rats (10/sex/group) were exposed to greater than 99% pure EBU for 13 weeks at the following concentrations: 0, 50, 100, 200, 400, or 800 ppm (0, 147, 295, 590, 1180, or 2360 mg/cu.m) (NTP, 1988). The corresponding duration-adjusted values, based on exposure 6 hours/day, 5 days/week, were 0, 26, 53, 105, 211, or 421 mg/cu.m. Vapor was generated from the heated liquid and pumped directly into the exposure chamber. The animals were observed twice daily and weighed weekly. Histopathological examinations were performed on approximately 30 tissues from control animals and animals from the two highest concentration groups. The nasal turbinates were examined in all animals at all exposure levels. NTP procedures were followed in the preparation and examination of all tissues. A significant reduction in body weight was observed in rats exposed to 800 ppm EBU, but surviving rats exhibited no change in body weight compared with controls. Inflammation of the nasal turbinates was noted in rats exposed to 800 ppm (10/10 with acute rhinitis) but not in rats at lower concentrations. The inflammation was present in both the dorsal and lateral portions of the nasal cavity, affecting both the respiratory and olfactory epithelium. The NOAEL for these effects was 400 ppm, RGDR = 0.107, [NOAEL(HEC) = 22.6 mg/cu.m]. A lower concentration, 200 ppm, was a LOAEL in the chronic study described above (NTP, 1988).

Groups of 15 F344/N rats and B6C3F1 mice/sex were exposed to 0, 75, 150, or 600 ppm EBU (more than 99% pure) 6 hours/day 5 days/week for 13 weeks (Miller et al., 1981). These values correspond to 0, 221, 442, or 1770 mg/cu.m. Adjusted for exposure duration, these values become 0, 38, 79, or 317 mg/cu.m. The experimental protocol was the same as reported for the 2- week study (see next paragraph), except that 5 animals/sex/group were sacrificed after 4 weeks of exposure. No treatment-related deaths were observed. At the termination of the experiment, both sexes of mice exposed to the highest concentration had significant decreases in weight gain (7.6 g vs. 5.3 g for males and 7.5 g vs. 5.0 g for females). The authors noted alterations in the absolute and relative weights of several organs but did not present the data. Hematological data were not reported for the rats, and the authors claimed that no consistent treatment-related effects were observed. Male and female mice exhibited several statistically significant changes although there was no concentration-response relationship and the alterations were claimed to be within the range of historical controls (not reported). Clinical chemistry and urinalysis data were not reported, but the authors state that with the exception of reduced urine specific gravity in the 600-ppm male and female rats, no treatment-related effects were seen. In both the rats and mice exposed to 600 ppm EBU, histopathological examination revealed evidence of nasal mucosal irritation that consisted of flattening of the olfactory and respiratory epithelium with some focal thickening of the respiratory epithelium and increased numbers of inflammatory cells in the nasal cavity. No incidence rates were given in the study, nor was any information presented other than in narrative format concerning these effects. No effects were seen in the trachea or lungs. Other histopathological changes noted in rats and mice exposed to 600 ppm EBU included decreased hepatocellular size, decreased cell content in the cortex of the thymus gland, and myeloid hyperplasia in vertebral bone marrow (3 of 10 male rats). There were

no histopathologic observations in rats or mice in the two lower exposure groups that were considered treatment related. It is not clear why there is such a large discrepancy between this study, which supports a NOAEL at 150 ppm for both species, and the NTP subchronic studies, which clearly show effects in mice at 100 ppm as well as marked species differences in toxic response for the tissues of the upper respiratory tract. This study, however, does confirm that the nasal mucosa is the target of EBU toxicity in both rats and mice. A NOAEL of 150 ppm EBU is indicated for both rats and mice, [NOAEL(HEC) for female mice (RGDR = 0.122) = 9.6 mg/cu.m and for females rats (RGDR = 0.107) 8.4 mg/cu.m].

Miller et al. (1981) exposed F344 rats and B6C3F1 mice (5/sex/group) to 0, 400, 800, or 1600 ppm (0, 1180, 2360, or 4720 mg/cu.m) 99% EBU for a total of 9 days over a 2-week period. Exposures were for 6 hours/day, 5 days/week; the duration-adjusted concentrations were 0, 211, 422, or 845 mg/cu.m. The test atmosphere was analyzed 2-3 times/hour by infrared spectroscopy. Clinical observations were conducted daily, and body weights were monitored throughout the study. Hematology, clinical chemistry, and urinalyses were conducted. Complete necropsies were conducted at study termination and approximately 40 tissues were examined histopathologically including the nasal turbinates (number of sections not specified). All mice in the 1600-ppm group died by the third day of exposure, while the rats exposed to this concentration exhibited no clinical signs of toxicity, even though their body weight gain was significantly reduced (21% in males and 25% in females). Body weights of male rats exposed to 800 and 400 ppm EBU were not different from control values at the end of the experiment. Elevated mean white blood cell counts, a trend toward a decreased percentage of lymphocytes, and an increased percentage of neutrophils were reported for rats of both sexes exposed to 1600 ppm EBU (data not presented). Inflammatory and degenerative changes were noted in both the olfactory and respiratory portions of the nasal turbinates of both rats and mice exposed to 800 and 1600 ppm but not in animals exposed to 400 ppm. No treatment-related changes were seen in the trachea or lungs of these animals. Myeloid hyperplasia, noted in the vertebral bone marrow of most rats exposed to 1600 ppm and in some rats and mice exposed to 800 ppm EBU, may have been secondary to the nasal inflammation. No adverse effects were reported in the animals exposed to 400 ppm. The NOAEL(HEC) at this concentration based on the nasal tract (extrathoracic) effects is 21.9 mg/cu.m for both mice and rats (RGDR = 0.104 for both female rats and mice). The brevity of the exposure period (2 weeks) precludes the use of this study as the principal study.

Fourteen-day studies were also reported in the NTP (1988) study. Groups of five animals/sex of F344/N rats and B6C3F1 mice were exposed to 0, 400, 800, 1600, 3200, or 6400 ppm EBU (0, 1180, 2360, 4720, 9439, or 18878 mg/cu.m) 6 hours/day, 5 days/week (NTP, 1988). The corresponding duration-adjusted values are 0, 211, 421, 843, 1686, or 3371 mg/cu.m. Clinical observations were carried out twice daily and body weights were determined at study initiation, after 1 week, and at necropsy. Only a fraction of the animals were histologically examined. All

rats exposed to the two highest concentrations of EBU died before study termination, and 2/5 female rats exposed to 1600 ppm EBU died. A concentration-related reduction in body weight was observed in male and female rats exposed to 800 or 1600 ppm. Moderate multifocal pulmonary hemorrhage and acute suppurative rhinitis were observed in some of the rats exposed to 1600 ppm EBU. All mice exposed to concentrations greater than or equal to 1600 ppm EBU died, and 1/5 male mice exposed to 800 ppm EBU died before study termination. Body weight was reduced in the mice exposed to 800 ppm. The text of the study reports that moderate nephrosis was noted in 2/2 males exposed to 1600 ppm, and mild to slight nephrosis was seen in 2/2 males and 1/2 females exposed to 800 ppm EBU. This study indicates that mice are more sensitive than rats to the toxic effects of EBU, although other studies reported on below indicate that the rabbit may be more sensitive to the effects of this compound than either of these species.

The occurrence of systemic toxicity or cancer caused directly by EBU would be a function of dose to the target organ thereby implying systemic circulation of parent EBU. Data from NTP inhalation studies available for structural analogs of EBU indicate that the occurrence of neoplasms in organ systems other than the nasal cavity is present with the 2-carbon analog (ethylene oxide), but absent with the 3-carbon analog (propylene oxide) and the 4-carbon analog (EBU). One implication of this limited structure-response relationship is that EBU may not have significant distribution to remote sites under the conditions employed by NTP since only portal-of-entry neoplasia was noted. Thus, concern for remote or extrarespiratory effects, including reproductive and developmental effects, caused directly by EBU in these species is ameliorated. The studies discussed below may reflect this circumstance as no reproductive/developmental effects were noted. No evidence exists for EBU to exert toxicity through a secondary mechanism (e.g., through a metabolite). The limited metabolic data for EBU indicates that it is converted to a mercapturic acid via conjugation with glutathione (NTP, 1988).

Wolf (1961) exposed 4 groups of animals, each comprised of 10 rats (strain not indicated), 8 guinea pigs, and 2 rabbits per sex, to either 0 (2 groups of unexposed controls), 400 ppm, or 800 ppm of a blend of EBU isomers: 70% EBU, 15% 2,3-epoxybutane, and 10% isobutylene oxide (2-methyl-1,2-epoxypropane) for 7 hours/day. Each exposed group was subjected to 135-141 exposures over a period of 198 days (about 6.5 months). These exposure values correspond to 1180 and 2340 mg/cu.m. The duration-adjusted values (assuming 5 exposures days/week) would be 246 or 488 mg/cu.m. Growth and mortality records were kept for each group and the animals were observed frequently. At the end of the experiment, hematological exams were performed on the rats and rabbits, urinalysis on some of the same animals, and BUN on selected rats and rabbits. The lung, heart, kidneys, liver, spleen, and testes (but apparently no portion of the upper respiratory tract) were examined histologically. The lungs and other organs were weighed at the end of the experiment. Although mention is made of increased mortality among male rats, no data are given. Data presented showed that the growth of male, but not female, rats was adversely effected by the exposure, with an 11% weight deficit relative to controls at the higher

concentration. A similar growth depression (12%) was noted for male guinea pigs at this same concentration. Relative lung weights were increased in both sexes of rats and guinea pigs at 800, but not at 400, ppm. Erratic results from the few rabbits prevented the authors from making any claims about effects in those exposed to the higher concentration, although those exposed at the lower concentration were not different from the controls in the parameters noted. No adverse effects were reported at the 400-ppm exposure level indicating this concentration to be a NOAEL. Based on the increased lung weights (a thoracic effect, RGDR for female rats = 2.23), [NOAEL(HEC) = 548 mg/cu.m].

Sikov et al. (1981) exposed groups of 38-45 Wistar rats to 0, 250, or 1000 ppm EBU (more than 99% pure) 7 hours/day, 5 days/week for a 3 week pregestational period; or for 7 hours/day on gestation days 1-19; or for a combination of pregestational and gestational exposure. The test atmosphere was analyzed using a gas chromatograph. Analysis of the test material showed that none of the other isomeric butylene oxides were present in concentrations greater than 0.1%. Standard procedures were used to examine the fetuses on day 21 of gestation. One death occurred and body weight deficits of 10% relative to controls were noted in the dams exposed to 1000 ppm (n = 42) exposed during pregestation and gestation. Absolute and relative weights of the liver, lung, kidneys, or placenta of the exposed dams did not differ from the controls. Histopathology of the dams' lungs showed no exposure-related effects. No parameters of reproductive function were significantly altered by exposure to EBU, nor was fetal growth, viability, and development affected by these conditions of exposure. These results indicate that 250 ppm is a NOAEL for maternal toxicity (weight loss) and that the higher concentration of 1000 ppm (2950 mg/cu.m) is a free-standing NOAEL for fetotoxicity.

Sikov et al. (1981) also exposed groups of 24-49 New Zealand white rabbits to 0, 250, or 1000 ppm EBU (more than 99% pure) for 7 hours/day on gestation days 1-24. Standard techniques were used to evaluate the fetuses on gestation day 21. Death occurred in 14/24 rabbits exposed to 1000 ppm EBU and in 6/48 rabbits exposed to 250 ppm EBU. However, no effect on maternal weight gain was observed in the surviving animals. Absolute and relative weights of the liver, lung, kidneys, or placenta of the exposed animals did not differ from the controls. A decreased pregnancy rate was observed in the rabbits exposed to the highest concentration, although this finding may be confounded by the high mortality in this group. A decreased number of live fetuses per litter was observed in the two litters born to rabbits exposed to 1000 ppm EBU. Both a hypoplastic tail and unilateral renal agenesis was observed in 1 of the 8 surviving fetuses born to rabbits exposed to 1000 ppm EBU. No other embryotoxic or developmentally toxic effects were observed. Based on maternal effects, 250 ppm (737 mg/cu.m) is a frank effect level (FEL), as mortality was noted at this concentration. Evaluation of fetotoxicity in this study was not possible due to the limited data presented. Since extensive mortality was observed with rabbits but not rats (see above), rabbits could be considered as the more sensitive species of the two. The relative sensitivity to EBU of rabbits and female mice (NTP,1988), however, is not clear.

Pharmacokinetic studies were conducted in male F344 rats by Reitz et al. (1983) to determine the major routes and rates of clearance following a 6-hour exposure to either 50 or 1000 ppm EBU or gavage administration of 20 mg/kg EBU in corn oil. Results of these studies indicate that EBU is rapidly absorbed, metabolized, and eliminated following either inhalation or oral exposure. EBU was eliminated primarily as nonvolatile urinary metabolites in the urine or as expired carbon dioxide. Practically all of the administered EBU was recovered within the first 36 hours after exposure, with small amounts remaining in the carcass after 32 or 66 hours. The identity of the urinary metabolites was not determined. There were significant differences in the amount of radioactivity recovered in the urine and expired air between animals given C1-labeled EBU and uniformly labeled EBU. Exposure to C1-EBU resulted in the recovery of 40- 46% of the radioactivity in the urine whereas exposure to uniformly labeled EBU resulted in the recovery of only 12% of the radioactivity in the urine. The recovery of radioactivity in the expired air was less in animals exposed to C1-labeled EBU (27-34%) and more in animals exposed to uniformly labeled EBU (58%). The authors concluded that this labeling pattern indicates that the urinary metabolites contain only the label associated with the C1-atom of EBU and thus cannot be simple conjugates of EBU. There was no significant differences in the percentages of radioactivity recovered in the urine and expired air following exposure to either 50 or 2000 ppm, indicating that the pharmacokinetics of EBU are linear over this range of concentrations.

I.B.5. Confidence in the Inhalation RfC

Study — Medium
Database — Medium
RfC — Medium

Although the NTP (1988) chronic inhalation study was well-conducted, used an appropriate number of animals, chose well-spaced exposure levels, and provided a thorough histopathological examination of the respiratory tract, it did not establish a NOAEL and can be given no more than a medium confidence rating. The critical effect identified in this study, lesions of the nasal tract, is supported by the effects seen in rats in the subchronic NTP (1988) studies, as well as in subchronic studies in rats and mice (Miller et al., 1981; NTP, 1988). Female mice are more sensitive than males to EBU. The maternal effects seen in rabbits indicate that this species may be even more sensitive than female mice. The database is given a medium confidence rating because there is a chronic inhalation study in two species supported by subchronic inhalation studies in several species, and because inhalation studies are available on developmental effects. There are no multigenerational reproductive studies available. A medium confidence rating in the RfC follows.

I.B.6. EPA Documentation and Review of the Inhalation RfC

Source Document — This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation — None

Agency Work Group Review — 12/11/1991

Verification Date — 12/11/1991

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfC for 1,2-Epoxybutane (EBU) conducted in September 2002 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or (202)566-1676.

I.B.7. EPA Contacts (Inhalation RfC)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — 1,2-Epoxybutane (EBU)
CASRN — 106-88-7

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — 1,2-Epoxybutane (EBU)
CASRN — 106-88-7

VI.A. Oral RfD References

None

VI.B. Inhalation RfC References

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NTP (National Toxicology Program). 1988. Toxicology and carcinogenesis studies of 1,2-epoxybutane (CAS No. 106-88-7) in F344/N rats and B6C3F1 mice (inhalation studies). ISS NTP-TR-329, NIH/PUB-88-2585. Additional data on the subchronic studies were supplied by NTP and are included in the IRIS data file on this chemical.

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VI.C. Carcinogenicity Assessment References

None

VII. Revision History

Substance Name — 1,2-Epoxybutane (EBU)
CASRN — 106-88-7

Date	Section	Description
05/01/1992	I.B.	Inhalation RfC on-line
12/03/2002	I.B.6.	Screening-Level Literature Review Findings message has been added.

VIII. Synonyms

Substance Name — 1,2-Epoxybutane (EBU)
CASRN — 106-88-7
Last Revised — 05/01/1992

- 106-88-7
- 1,2-epoxybutane
- butane, 1,2-epoxy-
- butylene oxide
- ethylethylene oxide
- N-butene-1,2-oxide
- 1-butene oxide
- 1-butylene oxide
- 1,2-butene oxide
- 1,2-Butylene oxide
- 2-ethyloxirane